09/917,154
1-4
Att 10

WEST Search History

DATE: Tuesday, March 04, 2003

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ		
L9	L8 not l7	143	L9
L8	l1 with l2	157	L8
L7	l1 with l3 with L6	293	L7
L6	express$	715375	L6
L5	l1 with l3	1289	L5
L4	l1 with l2 with l3	13	L4
L3	(blood vessel) or vascula$ vein or venus or artery or arterial	99478	L3
L2	parenchym$	4875	L2
L1	polynucleotide or oligonucleotide or (nucleic acid) or dna or rna or plasmid	194054	L1

END OF SEARCH HISTORY

=> s dna or rna or plasmid!? or oligonucleotide!? or polynucleotide!? or (nucleic acid!?)
3 FILES SEARCHED...
L1 3289976 DNA OR RNA OR PLASMID!? OR OLIGONUCLEOTIDE!? OR POLYNUCLEOTIDE!? OR (NUCLEIC ACID!?)

=> s blood vessel!?

L2 105295 BLOOD VESSEL!?

=> s gene therapy

L3 89288 GENE THERAPY

=> s l1 and l2 and l3

L4 239 L1 AND L2 AND L3

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 161 DUP REM L4 (78 DUPLICATES REMOVED)

=> s l5 and py<1998
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L6 32 L5 AND PY<1998

=> d l6 ibib abs 1-32

L6 ANSWER 1 OF 32 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:35805 BIOSIS
DOCUMENT NUMBER: PREV199800035805
TITLE: Targeting ***gene*** ***therapy*** to cancer: A review.
AUTHOR(S): Dachs, Gabi U. (1); Dougherty, Graeme J.; Stratford, Ian J.; Chaplin, Dai J.
CORPORATE SOURCE: (1) Gray Lab., Mount Vernon Hosp., Northwood, Middlesex HA6 2JR UK
SOURCE: Oncology Research, (1997) Vol. 9, No. 6-7, pp. 313-325. ISSN: 0965-0407.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB In recent years the idea of using ***gene*** ***therapy*** as a modality in the treatment of diseases other than genetically inherited, monogenic disorders has taken root. This is particularly obvious in the field of oncology where currently more than 100 clinical trials have been approved worldwide. This report will summarize some of the exciting progress that has recently been made with respect to both targeting the delivery of potentially therapeutic genes to tumor sites and regulating their expression within the tumor microenvironment. In order to specifically target malignant cells while at the same time sparing normal tissue, cancer ***gene*** ***therapy*** will need to combine highly selective gene delivery with highly specific gene expression, specific gene product activity, and, possibly, specific drug activation. Although the efficient delivery or ***DNA*** to tumor sites remains a formidable task, progress has been made in recent years using both viral (retrovirus, adenovirus, adeno-associated virus) and nonviral (liposomes, Bene gun, injection) methods. In this report emphasis will be placed on targeted rather than high-efficiency delivery, although those would need to be combined in the future for effective therapy. To date delivery has been targeted to tumor-specific and tissue-specific antigens, such as epithelial growth factor receptor, c-kit receptor, and folate receptor, and these will be described in some detail. To increase specificity and safety of ***gene*** ***therapy*** further, the expression of the therapeutic gene needs to be tightly controlled within the target tissue. Targeted gene expression has been analyzed using tissue-specific promoters (breast-, prostate-, and melanoma-specific promoters) and disease-specific promoters (carcinoembryonic antigen, HER-2/neu, Myc-Max response elements, DF3/MUC). Alternatively, expression could be regulated externally with the use of radiation-induced promoters or tetracycline-responsive elements. Another novel possibility that will be discussed is the regulation of therapeutic gene products by tumor-specific gene splicing. Gene expression could also be targeted at conditions specific to the tumor microenvironment, such as glucose deprivation and hypoxia. We have concentrated on hypoxia-targeted gene expression and this report will discuss our progress in detail. Chronic hypoxia occurs in tissue that is more than 100-200 pm away from a functional blood supply. In solid rumors hypoxia is widespread both because cancer cells are more prolific than the invading endothelial cells that make up the ***blood*** ***vessels*** and because the newly formed blood supply is disorganized. Measurements of oxygen partial pressure in patients' tumors showed a high percentage of severe hypoxia readings (less than 2.5 mmHg), readings not seen in normal tissue. This is a major problem in the treatment of cancer, because hypoxic cells are resistant to radiotherapy and often to chemotherapy. However, severe hypoxia is also a physiological condition specific to tumors, which makes it a potentially exploitable target. We have utilized hypoxia response elements (HRE) derived from the oxygen-regulated phosphoglycerate kinase gene to control gene expression in human tumor cells in vitro and in experimental tumors. The list of genes that have been considered for use in the treatment of cancer is extensive. It includes cytokines and costimulatory cell surface molecules intended to induce an effective systemic immune response against tumor antigens that would not otherwise develop. Other inventive strategies include the use of internally expressed antibodies to target oncogenic proteins (intrabodies) and the use of antisense technology (antisense ***oligonucleotides*** , antigenes, and ribozymes). This report will concentrate more on novel genes encoding prodrug activating enzymes, so-called suicide genes (Herpes simplex virus thymidine kinase, Escherichia coli nitroreductase, E. coli cytosine deaminase, thymidine phosphorylase, cytochrome P450 isoforms, deoxycytidine kinase, and our initial work on the Clostridium acetobutylicum hydrogenase and flavodoxin), and their prospective prodrugs. Details of our work on placing the gene encoding cytosine deaminase under hypoxia control will be discussed.

L6 ANSWER 2 OF 32 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:1980 BIOSIS
DOCUMENT NUMBER: PREV199800001980
TITLE: Angiogenesis by ***gene*** ***therapy*** : A new horizon for myocardial revascularization.
AUTHOR(S): Lewis, Basil S. (1); Flugelman, Moshe Y.; Weisz, Anat; Keren-Tal, Iris; Schaper, Wolfgang
CORPORATE SOURCE: (1) Dep. Cardiol., Lady Davis Carmel Med. Cent., 7 Michal St., Haifa 34362 Israel
SOURCE: Cardiovascular Research, (***Sept., 1997***) Vol. 35, No. 3, pp. 490-497. ISSN: 0008-6363.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB The concept of therapeutic angiogenesis is based on the premise that the potential for vascular growth inherent in vascular tissue can be utilized to promote the development of new ***blood*** ***vessels*** under the influence of the appropriate growth factors. Direct application of growth factors of the fibroblast (acidic, basic fibroblast growth factor, FGF-5), endothelial (vascular endothelial growth factor) and other series has been effective in preliminary studies. Angiogenesis by gene transfer provides an attractive alternative, with the advantage that the protein may continue to be secreted for a longer period of time and that the gene may be targeted to specific tissues to enhance efficacy and reduce systemic side effects. Angiogenesis by gene transfer is currently under investigation using a variety of growth factors and a wide array of potential delivery systems. These include application of the gene as naked ***DNA*** or by viral vector in the proximal vessel by direct intravascular injection, interventional cardiologic techniques (hydrogel coating on balloon, double balloon system, stent implantation) or by direct application to adventitia, pericardium or ischemic tissue distal to the site of arterial obstruction. As our understanding of the molecular and genetic processes underlying angiogenesis increases, and as we examine

09/917154

=> s dna or rna or plasmid!? or oligonucleotide!? or polynucleotide!? or (nucleic acid!?)
3 FILES SEARCHED...
L1 3289976 DNA OR RNA OR PLASMID!? OR OLIGONUCLEOTIDE!? OR POLYNUCLEOTIDE!? OR (NUCLEIC ACID!?)

=> s (blood vessel!?) or vascular?
L2 1661148 (BLOOD VESSEL!?) OR VASCULAR?

=> s gene therapy
L3 89288 GENE THERAPY

=> s express?
2 FILES SEARCHED...
L4 3121978 EXPRESS?

=> s l3 or l4

3 FILES SEARCHED...
L5 3163383 L3 OR L4

=> s l1 and l2 and l5

L6 45028 L1 AND L2 AND L5

=> s l1 and l2 and l3 and l4
L7 1523 L1 AND L2 AND L3 AND L4

=> s parenchym?
L8 113782 PARENCHYM?

=> s l1 and l2 and l3 and l4 and l8
L9 12 L1 AND L2 AND L3 AND L4 AND L8

=> s extravascul?
L10 16978 EXTRAVASCUL?

=> s l8 or l10
L11 130398 L8 OR L10

=> s l1 and l2 and l3 and l4 and l11
L12 15 L1 AND L2 AND L3 AND L4 AND L11

=> s l1 and l2 and l4 and l11
L13 556 L1 AND L2 AND L4 AND L11

=> s blood vessel!?
L14 105295 BLOOD VESSEL!?

=> s l1 and l4 and l11 and l14
L15 115 L1 AND L4 AND L11 AND L14

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 54 DUP REM L15 (61 DUPLICATES REMOVED)

=> s l16 and py<1998
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L17 28 L16 AND PY<1998

=> dup rem l13

PROCESSING COMPLETED FOR L13
L18 334 DUP REM L13 (222 DUPLICATES REMOVED)

=> s l18 and py<1998

1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L19 139 L18 AND PY<1998

=> s l17 or l19

L20 139 L17 OR L19

=> d l20 ibib abs 1-139

L20 ANSWER 1 OF 139 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:79746 BIOSIS
DOCUMENT NUMBER: PREV199800079746
TITLE: T-cell intravascular lymphomatosis (angiotropic large cell lymphoma): Association with Epstein-Barr viral infection.
AUTHOR(S): Au, W. Y.; Shek, W. H.; Nicholls, J.; Tse, K. M.; Todd, D.;
Kwong, Y. L. (1)
CORPORATE SOURCE: (1) Univ. Dep. Med., Prof. Block, Queen Mary Hosp.,
Pokfulam Road, Hong Kong Hong Kong
SOURCE: Histopathology (Oxford), (***Dec., 1997***) Vol. 31, No. 6, pp. 563-567.
ISSN: 0309-0167.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Aims: Intravascular lymphomatosis (IVL) is a very rare non-Hodgkin's lymphoma characterized by proliferation of lymphoma cells in the ***vascular*** lumina without involvement of adjacent ***parenchymal*** tissue. IVL is predominantly of B-cell lineage, but occasional cases of T lineage IVL involving almost exclusively the skin have been described. A case of IVL that occurred initially in the epididymis of a patient with an antecedent nasopharyngeal carcinoma was studied to define the clinicopathological features associated with this unique presentation. Methods and results: This lymphoma was studied by standard histological and immunophenotyping methods. The results showed lymphoma cells confined within the ***blood*** ***vessels*** , which ***expressed*** leucocyte common antigen, and T-cell markers CD3 and UCHL-1. The T-cell origin of the IVL prompted investigations for an association with Epstein-Barr virus infection (EBV). In situ hybridization with digoxigenin-labelled anti-sense ***RNA*** probes to EBV encoded ***RNA*** (EBER) showed strong signals in the nuclei of virtually all of the lymphoma cells. Conclusions: EBV infection of the malignant cells was demonstrated by in-situ hybridization. This case suggests that T-cell IVL may be another EBV related human neoplasm. This observation will need to be validated by further studies.

L20 ANSWER 2 OF 139 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:47280 BIOSIS
DOCUMENT NUMBER: PREV199800047280
TITLE: Tissue-specific regulation of renal and cardiac atrial natriuretic factor gene ***expression*** in deoxycorticosterone acetate-salt rats.
AUTHOR(S): Ogawa, Tsuneo; Bruneau, Benoit G.; Yokota, Naoto; De Bold,
Mercedes L. Kuroski; De Bold, Adolfo J. (1)
CORPORATE SOURCE: (1) Univ. Ottawa Heart Inst. Res. Centre, Ottawa Civic
Hosp., 1053 Carling Ave., Ottawa, ON K1Y 4E9 Canada
SOURCE: Hypertension (Dallas), (***Dec., 1997***) Vol. 30, No. 6, pp. 1342-1347.
ISSN: 0194-911X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Atrial natriuretic factor (ANF) is ***expressed*** in several noncardiac tissues where it may have an autocrine or paracrine function. Such function may be expected of locally synthesized ANF in the renal ***parenchyma*** . Previous investigations of the existence of ANF mRNA in the renal ***parenchyma*** have yielded conflicting results. The investigations reported here were designed to detect and measure ANF mRNA in normal rats and in rats subjected to a deoxycorticosterone acetate (DOCA)-salt treatment schedule known to strongly activate cardiac ANF gene ***expression*** . The ***expression*** of the renal ANF gene was

=> s (gene or genes)(2n)(deliver or delivery or delivered)
L1 17448 (GENE OR GENES)(2N)(DELIVER OR DELIVERY OR DELIVERED)

=> s gene!?(2n)transfer?

4 FILES SEARCHED...
L2 29658 GENE!?(2N) TRANSFER?

=> s l1 or l2
L3 46187 L1 OR L2

=> s blood vessel!?
L4 105295 BLOOD VESSEL!?

=> s l3 and l4
L5 187 L3 AND L4

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 100 DUP REM L5 (87 DUPLICATES REMOVED)

=> s l6 and py<1998
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L7 27 L6 AND PY<1998

=> d l7 ibib abs 1-27

L7 ANSWER 1 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:35805 BIOSIS
DOCUMENT NUMBER: PREV199800035805
TITLE: Targeting gene therapy to cancer: A review.
AUTHOR(S): Dachs, Gabi U. (1); Dougherty, Graeme J.; Stratford, Ian J.; Chaplin, Dai J.
CORPORATE SOURCE: (1) Gray Lab., Mount Vernon Hosp., Northwood, Middlesex HA6 2JR UK
SOURCE: Oncology Research, (1997) Vol. 9, No. 6-7, pp. 313-325. ISSN: 0965-0407.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB In recent years the idea of using gene therapy as a modality in the treatment of diseases other than genetically inherited, monogenic disorders has taken root. This is particularly obvious in the field of oncology where currently more than 100 clinical trials have been approved worldwide. This report will summarize some of the exciting progress that has recently been made with respect to both targeting the delivery of potentially therapeutic genes to tumor sites and regulating their expression within the tumor microenvironment. In order to specifically target malignant cells while at the same time sparing normal tissue, cancer gene therapy will need to combine highly selective ***gene*** ***delivery*** with highly specific gene expression, specific gene product activity, and, possibly, specific drug activation. Although the efficient delivery or DNA to tumor sites remains a formidable task, progress has been made in recent years using both viral (retrovirus, adenovirus, adeno-associated virus) and nonviral (liposomes, Bene gun, injection) methods. In this report emphasis will be placed on targeted rather than high-efficiency delivery, although those would need to be combined in the future for effective therapy. To date delivery has been targeted to tumor-specific and tissue-specific antigens, such as epithelial growth factor receptor, c-kit receptor, and folate receptor, and these will be described in some detail. To increase specificity and safety of gene therapy further, the expression of the therapeutic gene needs to be tightly controlled within the target tissue. Targeted gene expression has been analyzed using tissue-specific promoters (breast-, prostate-, and melanoma-specific promoters) and disease-specific promoters (carcinoembryonic antigen, HER-2/neu, Myc-Max response elements, DF3/MUC). Alternatively, expression could be regulated externally with the use of radiation-induced promoters or tetracycline-responsive elements. Another novel possibility that will be discussed is the regulation of therapeutic gene products by tumor-specific gene splicing. Gene expression could also be targeted at conditions specific to the tumor microenvironment, such as glucose deprivation and hypoxia. We have concentrated on hypoxia-targeted gene expression and this report will discuss our progress in detail. Chronic hypoxia occurs in tissue that is more than 100-200 pm away from a functional blood supply. In solid rumors hypoxia is widespread both because cancer cells are more prolific than the invading endothelial cells that make up the ***blood*** ***vessels*** and because the newly formed blood supply is disorganized. Measurements of oxygen partial pressure in patients' tumors showed a high percentage of severe hypoxia readings (less than 2.5 mmHg), readings not seen in normal tissue. This is a major problem in the treatment of cancer, because hypoxic cells are resistant to radiotherapy and often to chemotherapy. However, severe hypoxia is also a physiological condition specific to tumors, which makes it a potentially exploitable target. We have utilized hypoxia response elements (HRE) derived from the oxygen-regulated phosphoglycerate kinase gene to control gene expression in human tumor cells in vitro and in experimental tumors. The list of genes that have been considered for use in the treatment of cancer is extensive. It includes cytokines and costimulatory cell surface molecules intended to induce an effective systemic immune response against tumor antigens that would not otherwise develop. Other inventive strategies include the use of internally expressed antibodies to target oncogenic proteins (intrabodies) and the use of antisense technology (antisense oligonucleotides, antigenes, and ribozymes). This report will concentrate more on novel genes encoding prodrug activating enzymes, so-called suicide genes (Herpes simplex virus thymidine kinase, Escherichia coli nitroreductase, E. coli cytosine deaminase, thymidine phosphorylase, cytochrome P450 isoforms, deoxycytidine kinase, and our initial work on the Clostridium acetobutylicum hydrogenase and flavodoxin), and their prospective prodrugs. Details of our work on placing the gene encoding cytosine deaminase under hypoxia control will be discussed.

L7 ANSWER 2 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:449912 BIOSIS
DOCUMENT NUMBER: PREV199799749115
TITLE: Novel methods for adenovirus-mediated gene transfer to ***blood*** ***vessels*** in vivo.
AUTHOR(S): Ooboshi, Hiroaki; Rios, C. David; Heistad, Donald D. (1)
CORPORATE SOURCE: (1) Dep. Intern. Med., Univ. Iowa Coll. Med., Iowa City, IA 52242 USA
SOURCE: Molecular and Cellular Biochemistry, (1997) Vol. 172, No. 1-2, pp. 37-46. ISSN: 0300-8177.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Adenovirus-mediated gene transfer is a promising method for studies of vascular biology and potentially for gene therapy. Intravascular approaches for gene transfer to ***blood*** ***vessels*** in vivo generally require interruption of blood flow and have several limitations. We have used two alternative approaches for gene transfer to ***blood*** ***vessels*** in vivo using perivascular application of vectors. First, replication-deficient adenovirus expressing nuclear-targeted bacterial beta-galactosidase was injected into cerebrospinal fluid via the cisterna magna of rats. Leptomeningeal cells over the major arteries were efficiently transfected, and adventitial cells of large vessels and smooth muscle cells of small vessels were occasionally stained. When viral suspension was injected with the rat in a lateral position, the reporter gene was expressed extensively on the ipsilateral surface of the brain. Thus, adenovirus injected into cerebrospinal fluid provides gene transfer in vivo to cerebral ***blood*** ***vessels*** and, with greater efficiency, to perivascular tissue. Furthermore, positioning of the head may 'target' specific regions of the brain. Second, vascular ***gene*** ***delivery*** was accomplished by perivascular injection of virus in peripheral vessels. Injection of the adenoviral vector within the periarterial sheath of monkeys resulted in gene transfer to the vessel wall that was substantial in magnitude although limited to cells in the adventitia. Approximately 20% of adventitial cells expressed the transgene, with no gene transfer to cells in the intima or media. These approaches may provide alternative approaches for gene transfer to ***blood*** ***vessels*** , and may be useful for studies of

WEST

Print Selection

Help | Clear | Cancel | Print | Print First Page

Select?	Document ID	Section(s)	Page(s)	# Pages to print	Database
☑	5885968	all	all	35	USPT,PGPB,JPAB,EPAB,DWPI
☑	5676954	all	all	13	USPT,PGPB,JPAB,EPAB,DWPI
☑	5328470	all	all	20	USPT,PGPB,JPAB,EPAB,DWPI
☑	US5026558A	all	all	9	USPT,PGPB,JPAB,EPAB,DWPI

Building cm1 **Room** 11e14 **Printer** gbldptr

Main Menu | Logout

WEST

Print Selection

Help | Clear | Cancel | Print | Print First Page

Select?	Document ID	Section(s)	Page(s)	# Pages to print	Database
☑	6515199	all	all	8	USPT,PGPB,JPAB,EPAB,DWPI
☑	5855910	all	all	14	USPT,PGPB,JPAB,EPAB,DWPI
☑	5498539	all	all	23	USPT,PGPB,JPAB,EPAB,DWPI
☑	5320962	all	all	19	USPT,PGPB,JPAB,EPAB,DWPI
☑	5208019	all	all	11	USPT,PGPB,JPAB,EPAB,DWPI
☑	5593974	all	all	24	USPT,PGPB,JPAB,EPAB,DWPI
☑	5631237	all	all	45	USPT,PGPB,JPAB,EPAB,DWPI
☑	5698531	all	all	13	USPT,PGPB,JPAB,EPAB,DWPI
☑	JP02001213806A	all	all	N/A	USPT,PGPB,JPAB,EPAB,DWPI

Building cm1 **Room** 11e14 **Printer** gbldptr

Main Menu | Logout